Answer 1:

Bibliographic Information

Methylator resistance mediated by mismatch repair deficiency in a glioblastoma multiforme xenograft. Friedman, Henry S.; Johnson, Stewart P.; Dong, Qing; Schold, S. Clifford; Rasheed, B. K. Ahmed; Bigner, Sandra H.; Ali-Osman, Francis; Dolan, Eileen; Colvin, O. Michael; Houghton, Peter; Germain, Glen; Drummond, James T.; Keir, Stephen; Marcelli, Susan; Bigner, Darell D.; Modrich, Paul. Department of Pediatrics, Duke University Medical Center, Durham, NC, USA. Cancer Research (1997), 57(14), 2933-2936. Publisher: American Association for Cancer Research, CODEN: CNREA8 ISSN: 0008-5472. Journal written in English. CAN 127:185456 AN 1997:477772 CAPLUS (Copyright (C) 2008 ACS on SciFinder (R))

Abstract

A methylator-resistant human glioblastoma multiforme xenograft, D-245 MG (PR), in athymic nude mice was established by serially treating the parent xenograft D-245 MG with procarbazine. D-245 MG xenografts were sensitive to procarbazine, temozolomide, N-methyl-N-nitrosourea, 1,3-bis(2-chloroethyl)-1-nitrosourea, 9-aminocamptothecin, topotecan, CPT-11, cyclophosphamide, and busulfan. D-245 MG (PR) xenografts were resistant to procarbazine, temozolomide, N-methyl-N-nitrosourea, and busulfan, but they were sensitive to the other agents. Both D-245 MG and D-245 MG (PR) xenografts displayed no O6-alkylguanine-DNA alkyltransferase activity, and their levels of glutathione and glutathione-S-transferase were similar. D-245 MG xenografts expressed the human mismatch repair proteins hMSH2 and hMLH1, whereas D-245 MG (PR) expressed hMLH1 but not hMSH2.

Answer 2:

Bibliographic Information

An experimental study of xenografted human gliomas. Bloom, H. J. G.; Bradley, N. J.; Davies, A. J. S.; Richardson, S. G. Inst. Cancer Res., R. Marsden Hosp., London, UK. Editor(s): Walker, Michael D.; Thomas, David G. T. Biol. Brain Tumour, Proc. Int. Symp., 2nd (1986), Meeting Date 1984, 415-21. Publisher: Nijhoff, Boston, Mass CODEN: 55JOAH Conference written in English. CAN 106:113248 AN 1987:113248 CAPLUS (Copyright (C) 2008 ACS on SciFinder (R))

Abstract

Chemotherapy of human astrocytomas growing as s.c. xenografts in mice revealed that the Grade III tumors were relatively insensitive but that agents such as procarbazine [671-16-9] and BCNU [154-93-8] were capable of procuring significant growth delay when used to treat Grade IV tumors. Against Grade IV tumors growing intracerebrally, procarbazine and BCNU were also effective agents but not in all animals. In a preliminary series of expts. surgical resection was shown to augment tumor growth rates which could be reduced by perioperative adjuvant chemotherapy.

Answer 3:

Bibliographic Information

Xenografts in pharmacologically immunosuppressed mice as a model to test the chemotherapeutic sensitivity of human tumors. Floersheim, G. L.; Bieri, A.; Chiodetti, Nicole. Zent. Lehre Forsch., Kantonssp., Basel, Switz. International Journal of Cancer (1986), 37(1), 109-14. CODEN: IJCNAW ISSN: 0020-7136. Journal written in English. CAN 104:81665 AN 1986:81665 CAPLUS (Copyright (C) 2008 ACS on SciFinder (R))

Abstract

A human tumor xenograft model using pharmacol. immunosuppressed mice was assessed for its suitability to test preclinically the sensitivity of colorectal carcinomas, bone sarcomas and melanomas against anticancer agents. Beside ionizing radiation, 14 cytotoxic

drugs including 5-fluorouracil (5-FU) [51-21-8], dimethylmyleran (DMM) [55-93-6], cytosine arabinoside [147-94-4], cyclophosphamide [50-18-0], melphalan [148-82-3], mitomycin C [50-07-7], adriamycin [23214-92-8], bleomycin [11056-06-7], etoposide [33419-42-0], vinblastine [865-21-4], cisplatin [15663-27-1], procarbazine [671-16-9], DTIC [4342-03-4], and BCNU [154-93-8] were assayed. Ionizing radiation, 5-FU and DMM were also applied at LDs followed by bone-marrow rescue high-dose therapy. Four colon carcinomas responded poorly to most of the agents but one tumor displayed marked sensitivity to BCNU. LDs of radiation, 5-FU and DMM and cyclophosphamide and by an osteosarcoma to the latter drug. No strong effects were seen against melanomas. LDs of DMM induced the best regression of one colon carcinoma. In general, the superiority of high-dose therapy for solid human tumors compared to maximally tolerated doses was demonstrated. Individual carcinomas of the same type displayed different drug sensitivity.

Answer 4:

Bibliographic Information

Chemotherapy and radiation therapy of human medulloblastoma in athymic nude mice. Friedman, Henry S.; Schold, S. Clifford, Jr.; Varia, Mahesh; Bigner, Darell D. Med. Cent., Duke Univ., Durham, NC, USA. Cancer Research (1983), 43(7), 3088-93. CODEN: CNREA8 ISSN: 0008-5472. Journal written in English. CAN 99:63962 AN 1983:463962 CAPLUS

Abstract

The human medulloblastoma cell line TE-671 was grown s.c. and intracranially in athymic nude mice. Tumor-bearing animals treated with chemotherapeutic agents or radiation were compared to untreated tumor-bearing controls. Tumors growing s.c. were sensitive to cyclophosphamide [50-18-0] and vincristine [57-22-7] with growth delays in duplicate trials of 15.8/16.5 and 12.9/15.0 days, resp. These tumors were minimally responsive to the 2,5-bis(1-aziridinyl)-3,6-dioxodiethyl ester of 1,4-cyclohexadiene-1,4-dicarbamic acid [57998-68-2] and cis-diamminedichloroplatinum II [15663-27-1] and unresponsive to methotrexate [59-05-2], NSC 351521 [72732-56-0], NSC 409962 [154-93-8], and procarbazine [671-16-9]. Radiation therapy with 2500 or 1500 rads as a single fraction produced a marked response, with growth delays of 39.5 and 21.1 days, resp. Cyclophosphamide produced a significant increase in the median survival of mice with intracranial tumors. Vincristine produced a minimal increase in the median survival while no response was seen to the 2,5-bis(1-aziridinyl)-3,6-dioxodiethyl ester of 1,4-cyclohexadiene-1,4-dicarbamic acid at the dose level and schedule tested. This model system will allow further anal. of the therapeutic sensitivity of human medulloblastoma to other agents or combined-modality regimens.

Answer 5:

Bibliographic Information

Human brain tumor xenografts in nude mice as a chemotherapy model. Houchens, David P.; Ovejera, Artemio A.; Riblet, Sylva M.; Slagel, Donald E. Battelle Mem. Inst., Columbus, OH, USA. European Journal of Cancer & Clinical Oncology (1983), 19(6), 799-805. CODEN: EJCODS ISSN: 0277-5379. Journal written in English. CAN 99:63770 AN 1983:463770 CAPLUS

Abstract

Two human brain tumors which were previously established in nude mice were used to det. antitumor efficacy of various therapeutic agents. These tumors were a medulloblastoma (TE-671) and a glioma (U-251) with mass-doubling times of 3.5 and 5.5 days, resp., as s.c. implants in nude mice. Intracranial tumor challenge was accomplished by inoculating tissue culture-grown cells of either tumor into the right cerebral hemisphere to a depth of 3 mm. Groups of mice which had been inoculated with tumor were treated with various doses and schedules of antineoplastic compds. by the i.p. route. A new drug (rapamycin [53123-88-9]) was very effective against the U-251 tumor. This model system should prove valuable in assessing the effects of various chemotherapeutic modalities against brain tumors.

Answer 6:

Bibliographic Information

In vitro sensitivity of human melanoma xenografts to cytotoxic drugs. Correlation with in vivo chemosensitivity. Tveit, Kjell Magne; Fodstad, Oeystein; Olsnes, Sjur; Pihl, Alexander. Norwegian Cancer Society, Norsk Hydro's Inst. Cancer Res., Oslo, Norway. International Journal of Cancer (1980), 26(6), 717-22. CODEN: IJCNAW ISSN: 0020-7136. Journal written in English. CAN 94:76807 AN 1981:76807 CAPLUS

Abstract

Single-cell suspensions prepd. from 5 human melanomas, grown serially as xenografts in athymic nude mice, were exposed in vitro to increasing concns. of dacarbazine [4342-03-4], CCNU [13010-47-4], procarbazine [671-16-9], vinblastine [865-21-4], and the cancerostatic lectins abrin and ricin. The in vitro chemosensitivity of the cells, as measured by the drug concns. required to inhibit colony formation in soft agar by 50%, was correlated with the growth delay of the xenografts in vivo, previously obsd. after treatment of the animals with maximal tolerable doses of the same drugs. For each drug, the in vitro sensitivity of the different xenografts was strongly correlated with their response in vivo. Apparently, the soft agar test, as carried out here, adequately reflects the relative sensitivity of the xenografts in vivo. The data indicate that human xenografts may be used to develop quant. in vitro chemosensitivity tests.